

Provisional Conclusions.—There exists in children a non-congenital form of diffuse cerebral sclerosis in which the cortical layers of the brain are more specially affected, and which is distinguished from the other forms by its appearance in healthy children, either without known cause or after traumata, by the steadily progressive character of its symptoms and by the especial prominence of the gradually increasing *dementia*, which finally reaches an extreme degree without a corresponding loss of motor power, and while the sensation is comparatively unaffected.

[TO BE CONTINUED.]

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, May 6, 1890.

THE President, DR. LANDON CARTER GRAY, in the chair.

Dr. LANDON CARTER GRAY read a paper, entitled: "Can We Diagnose Hyperæmia and Anæmia of the Brain and Cord?" After citing the views of such recent writers as Gowers, Strümpell, Liebermeister, Ross, Seeligmüller and Hammond, and showing that they all, with the exception of the one last named, spoke with considerable skepticism of the possibility of diagnosis, the writer then passed in review the symptoms upon which the diagnosis is supposed to be made. Of these cephalgia, flushing and pallor of the face, vertigo, tinnitus aurium, insomnia, delirium, were, he said, general symptoms which can only derive diagnostic value from their associated symptoms. Slight retinal changes of vascularity are almost impossible to differentiate from the normal, as every well-informed ophthalmologist knows, and more marked retinal changes, such as neuro-retinitis, are indicative of organic intracranial disease. Myosis and motor and sensory paralysis have never yet been shown to have been caused by simple hyperæmia or anæmia of the brain. If the symptoms of apoplexy, paralysis, convulsions, a soporific condition, mania and aphasia, which Dr. Hammond states to be indicative of cerebral hyperæmia and anæmia are symptoms of organic intracranial disease, and the pathological data which are given by Dr. Hammond, such as enlarged and dilated blood-vessels, increase in consistence and density of the white matter, red or violet hue of the gray matter, subarachnoid or ventricular or choroid effusion, hematine, the *l'état criblé*

of Calmeil, miliary aneurisms, certainly show the existence of such organic disease as pronounced as in many cases of general paresis of the insane or in the severer cases of chorea or certain cases of intracranial syphilis. There is no proof that spinal anæmia or hyperæmia can be diagnosed whilst the symptoms given by many authors, on the one hand, are either too vague for differentiation from many slight affections, or, on the other hand, that of actual organic myelitis. Many diseases in the past, when they were poorly understood, were thought to be due to hyperæmia or anæmia of the brain or cord, such as meningitis, general paresis, porencephalitis, cerebral and spinal syphilis, disseminated sclerosis, disease of the medulla oblongata, myelitis of the anterior horns of the spinal cord, central and transverse myelitis, syringo-myelitis, progressive muscular atrophy, acute ascending or Landry's paralysis, locomotor ataxia, especially in its early stages, many forms of neuritis, focal disease of the brain in the light of our modern knowledge of localization, lateral sclerosis, many forms of lithæmia and neurasthenia. For these reasons the author does not believe that hyperæmia or anæmia of the brain or cord can be diagnosticated by means of the symptoms alone. If there are evidences of some intracranial disturbance, such as headache, delirium, vertigo, tinnitus aurium, insomnia, flushing or pallor of the face, and with these some concomitant conditions, making it reasonable to suppose that there might exist congestion or anæmia of the brain or cord, such a diagnosis might then be probable. Thus certain cardiac conditions, lesions of the great vessels of the chest and abdomen and thorax, certain forms of hepatic disease, tumors making pressure upon these great vessels, leucocythemia, intense mental complications or exposure to great heat; these conditions associated with the general symptoms might make it probable that the general symptoms were due to hyperæmia or anæmia, as the case might be, of the brain, but the writer does not believe that there are any pathological observations on record that would warrant a diagnosis of spinal anæmia or hyperæmia.

DISCUSSION.

Dr. W. R. BIRDSALL said he must admit that he had reached about the same conclusions as the reader of the address; that it was next to impossible to determine between the conditions of hyperæmia and anæmia by the nervous symptoms alone. There were, however, times when it was undoubtedly possible to recognize these states

from the general condition of the patient's system and from the course and character of the disease. Still, he thought there existed a great deal of confusion as to how the terms should be used. For instance, the term *anæmia* was often employed to express a mere malnutrition of the part. This application would be inappropriate in reference to the sense in which the President had discussed the subject. If it were a question whether the organs were supplied with too much or too little blood, of course variations in the quality of the blood must make considerable difference in the resulting symptoms, but he failed to appreciate how such symptoms could throw any light on the question. He believed that a large part of the symptoms which were brought forward as evidence of *hyperæmia* and *anæmia* were really conditions of disease due to malnutrition of the parts from the presence of poisonous substances in the blood, and not necessarily evidence of a defective or excessive supply of blood.

Dr. W. M. LESZYNSKY said that some years ago it had been the rule to diagnosticate cerebral *hyperæmia* upon the lines laid down by Dr. Hammond. The correctness of the diagnoses so made was assumed from the results of the results of the treatment. Patients in whom such symptoms were supposed to be developing were treated with bromides and ergot, because these drugs were thought to act directly upon the blood-vessels and the cerebral circulation. Many patients were benefited, and the diagnosis was therefore considered to be correct. The conclusion had, however, not been sustained. Investigation had demonstrated also that the condition of the retinal blood-vessels could not be taken as wholly indicative of the state of the circulation in the brain.

Dr. MARY PUTNAM JACOB remarked that many of the clinical symptoms which were most nearly associated with *anæmia* of the brain and which implied malnutrition of the cortex were unattended by any symptoms of general *anæmia*. The question of *anæmia*, therefore, so far as the brain was concerned, was complicated by the possibility of there being simply a vaso-motor spasm in the brain, which would diminish the blood supply and yet give no impression of general *anæmia*.

Dr. M. ALLEN STARR expressed his hearty agreement with the statements and conclusions of the writer of the paper. He was perfectly satisfied as to the impossibility of diagnosing between cerebral *anæmia* and *hyperæmia*. To pretend to do this was merely an evidence of incompetency on the part of the physician.